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# Environmental Health Disparities: A Framework Integrating Psychosocial and Environmental Concepts

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**Abbreviations:** EDP = exposure disease paradigm

**Outline of section headers:**

Abstract

Introduction

Health Disparities and the Environment

The Stress-Exposure Disease Framework

- The Exposure Disease Paradigm
- Race and Residential Location
- Environmental Burdens: Environmental Justice
- Structural Factors
- Community Stressors
  - Health effects of stress
  - Types of community stressors
  - Racial disparities in exposure to stressors
- Neighborhood Resources
- Community stress

Future Directions

Conclusion

**Abstract:**

Although it is often acknowledged that social and environmental factors interact to produce racial and ethnic environmental health disparities, it is still unclear how this occurs. Despite continued controversy, the environmental justice movement has provided some insight by suggesting that disadvantaged communities face greater likelihood of exposure to ambient hazards. The exposure disease paradigm has long suggested that differential "vulnerability" may modify the effects of toxins on biological systems. However, relatively little work has been done to specify whether racial and ethnic minorities may have greater vulnerability than majority populations, and further, what these vulnerabilities may be. We suggest that psychosocial stress may be the vulnerability factor that links social conditions with environmental hazards. Psychosocial stress can lead to acute and chronic changes in the functioning of body systems (e.g. immune) and also lead directly to illness.

We present a multidisciplinary framework integrating these ideas. We argue that residential segregation leads to differential experiences of community stress, exposure to pollutants, and access to community resources. When not counterbalanced by resources, stressors may lead to heightened vulnerability to environmental hazards.

## Introduction

The elimination of disparities in environmental health requires attention to both environmental hazards and social conditions (EPA, 2003). However, there are two major challenges implicit in this statement. First, is to understand how social processes may interrelate with environmental toxins. Second, is to understand why some groups experience greater illness compared to other groups. Our purpose of this article is to provide a multidisciplinary framework that addresses both issues.

We extend the work of Sexton and colleagues (1993), who documented how the exposure-disease paradigm could explain variation in the health of disadvantaged populations. Implicit in their framework is the idea that disadvantaged populations encounter greater susceptibility to environmental hazards. However, it is unclear what these susceptibility factors might be.

We suggest that psychosocial stress is a key component of differential susceptibility. Stressors, when not ameliorated by resources, may directly lead to health disparities. Additionally, stressors may amplify the effects of toxins. Residential segregation may be one important reason why communities differ in these exposures (Massey and Denton, 1993).

Our framework is built on an ecological perspective, suggesting that health disparities result not only from individual factors, but also from factors operating at multiple levels (Bronfenbrenner 1989; Sallis and Owen 1997). Reinvigoration in ecological approaches has paralleled the development of statistical techniques of multi-level modeling (e.g. hierarchical linear models), an appreciation that ecological factors

may not necessarily lead to the ecological fallacy, and a renewed interest in the role of context in health promotion (Diez-Roux 2000; Green and Kreuter 1999)

## Health Disparities and the Environment

Disparities exist for many health outcomes, including cancer, cardiovascular disease, diabetes, and mortality (U.S. Department of Health and Human Services 2000). Although there has been a national decrease in disparities between 1990-1998 (Keppel et al. 2002), some regions have reported an increase in disparities during the same period (Margollos, et al, 2004).

Environmental conditions are believed to play an important role in producing and maintaining health disparities (Lee 2002; Sexton 2000). Minority neighborhoods tend of have higher rates of mortality, morbidity, and health risk factors compared to white neighborhoods, even after accounting for economic and other characteristics (Cubbin et al. 2001; Geronimus 2000).

## The Stress-Exposure-Disease Framework

The Stress-Exposure-Disease Framework (Figure 1) provides a conceptual framework from which to understand the relationship between race, environmental conditions and health. It extends Sexton's (1993) framework by: (1) explicitly hypothesizing that residential segregation is a major reason why "race" is important; (2) incorporating an ecological, or multi-level perspective; (3) arguing that racial variation in stressors may account for differences in vulnerability to health risks.

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Figure 1 about here
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Reflecting the multi-level approach, the top panel of Figure 1 emphasizes community processes, while the bottom panel displays individual mechanisms. The tan shading reflects the Exposure-Disease Paradigm. To simplify our presentation, we dichotomize between individual and community processes. However, many processes are interrelated. For example, community wealth is party a function of individual wealth (e.g. when individuals contribute to the tax base) and individual wealth is also partly determined by community wealth (e.g. when rising property values benefit individual homeowners).

The framework shows that ethnicity is highly correlated with residential location, with minorities and whites often living segregated from one another. Differential residential location comes with differential exposure to health risks. In particular, neighborhood stressors and pollution sources create adverse health conditions, which are counterbalanced by neighborhood resources. Structural factors help determine the boundaries from which health promotion is possible and partially determine the contemporary state of stressors, resources, and pollution in a community. When community stressors and pollution sources outweigh neighborhood resources, levels of community stress manifest or increase. Community stress is a state of ecological vulnerability that may translate into individual stressors, which in turn, may lead to individual stress. Individual stress may then make individuals more vulnerable to illness when they are exposed to environmental hazards. Further, compromises in individual and community health may further weaken community resources, leading to a vicious cycle. Hence, we include in our framework a return loop from health back to stress.

As an example, zoning policies and tax incentives (structural factors), may encourage the entry of new pollutant industries. The increase in pollutants may lead to economic and social uncertainty (stressors) by driving down local property values, increasing the flight of jobs, and germinating a climate of uncertainty and fear. Neighborhood organizations (resources) may not be able to counterbalance these effects, leading to a state of community vulnerability (community stress). Community level vulnerability may in turn, may translate to individual vulnerability, such as when individuals are terminated from work or when individuals become anxious about perceived toxic exposures. When personal coping resources do not adequately counterbalance these external insults, individual stress and illness may result. Individual illness, in turn, may lead to further individual vulnerability, such as by reducing the ability to exercise. Additionally, individuals may affect their communities, such as when disaffected individuals cease participating in neighborhood organizations. Health disparities may arise because minorities are segregated into neighborhoods with high levels of community stress.

We do not explicitly examine the issue of genetic susceptibility in this framework for three reasons. First, we focus on factors that are amenable to policy change and social action. Second, genetic susceptibility is partly subsumed in the exposure disease paradigm because they are presumed to partially determine one's ability to defend against hazards. Third, while genetics are important in the etiology of many illnesses, it is likely that genetic factors do not explain racial health disparities (Cooper 1984; Cooper et al. 2003; Garte 2002; LaVeist 1994). It is often acknowledged that race is a social construct. What that means is that racial groups are not inherent biological

taxons, but represent societally defined categories during a particular point in history and place. For example, prior to 1989, the child of a black father and a white mother would be classified as black, but after 1989, the same child would be classified as white (LaVeist, 1994). Further, should the child have been born in Brazil, rather than the U.S., she would have been classified as Mulatto. Thus, racial designations are the product of social consensus and public policy, rather than biology *per se*.

Additionally, "genetically identified" groups tend to correlate poorly with socially identified groups because there is more genetic variation within than between groups (Lewontin 1982; Mountain and Cavalli-Sforza 1997; see Garte, 2002 for review). For example, genetic differences between any two Italians appear five-fold greater than the difference between an Italian and a Japanese, African, or New Guinean (Mountain and Cavalli-Sforza 1997). Observations such as these have led Cooper and colleagues (2003) to conclude, "Race… has not shown to provide a useful categorization of genetic information about the response to drugs, diagnosis, or causes of disease."

We now review the science that informs this framework, beginning with the exposure disease paradigm.

**The Exposure Disease Paradigm**

The Exposure-Disease Paradigm (EDP) is a well-known model that shows how environmental toxins might cause disease (Lioy 1990; National Research Council 1987; National Research Council 1991b; National Research Council 1991a). It is a continuum that includes the emission of a contaminant from a source through human exposure to the occurrence of a health effect.

Susceptibility/vulnerability intersects the continuum, increasing or decreasing resistance to absorption or and/or effect from toxins. The term susceptibility/vulnerability has been used broadly to cover both biological and non-biological factors, including genetic predisposition, pre-existing health conditions, and social conditions. The exact susceptibility/vulnerability factors and their pathways intersecting along the EDP are not well understood. We argue later that community and individual stress is one type of susceptibility factor.

Race and Residential Location

Segregation, the spatial separation of the residences of racial groups from one another, has persisted for many decades (Massey and Denton 1993; Iceland et al. 2002) . Table 1 shows the segregation of Blacks, Hispanics and Asians compared to whites from 1980 to 2000 for metropolitan areas, as measured with the index of dissimilarity (U.S. Census, 2004). Scored from 0-100, a given value of the index indicates the percent of that group who would have to move to integrate the metro area.

Segregation with whites are highest for African Americans, followed by Hispanics, Aisan Americans and Native Americans. In the average U.S. metropolis in the year 2000, about two-thirds of blacks (or whites) would have to move to another neighborhood in order to desegregate that metropolis.

Black-white and Native American-White segregation has declined since the 1980's, but segregation levels for Hispanics and Asians have remained stable. Further, most of the decline in black-white segregation has occurred in metro areas with the fewest numbers of blacks (Logan 2003).

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Table 1 about here
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The causes of segregation are still debated. Some have suggested that segregation is an artifact of broader shifts in the economy – including the decline of manufacturing jobs and suburbanization – that have left behind a cadre of the poor that are disproportionately racial minorities (Wilson 1987; Wilson 1996). Others have postulated that segregation results from personal preferences of homebuyers to cluster together (Schelling 1971). Most research has argued that segregation results from institutionalized discriminatory practices in the housing market (e.g. mortgage redlining, racialized "steering") that persists to the current day (Massey and Denton 1993; Meyer 2000; Munnell et al. 1996; Squires 1994).

Some evidence suggests that the mechanisms for segregation vary by ethnic group and region, but most ethnic groups have encountered discriminatory treatment historically and contemporaneously (Squires 1994; Feagin 2003; Krieger et al. 1993; Williams 1997a). For example, a recent audit study suggested that consistent adverse treatment in homebuying was similar for Asian American and African American homebuyers, with one in five potential homebuyers disfavored compared to whites (Turner et al. 2003). The causes of segregation notwithstanding, it is clear that neighborhoods do cluster on the basis of race and ethnicity.

Studies have reported that segregation is associated with numerous outcomes, including infant mortality (LaVeist 1989; LaVeist 1993; Haynaztka 2002), adult mortality (Polednak 1991;1996; Kindig et al. 2002; Hart et al. 1998), tuberculosis (Acevedo-Garcia 2003), homicide (Peterson and Krivo 1993; Peterson and Krivo 1999), teenage

childbearing (Sucoff and Upchurch 1998), exposure to tobacco and alcohol advertising (Alaniz 1998; Luke et al. 2000; U.S. Department of Health and Human Services 1998), and increased exposure to air pollution (Lopez 2002).

Segregation may thus be one critical link between race and environmental health disparities because racial groups, on average, occupy different residential areas. This may lead to differential exposure to health risk factors as well as differential access to resources. Segregation is multi-factorial, often conceptualized around five dimensions (Massey and Denton 1988, 1993; Avacedo-Garcia 2000). *Evenness*, the dimension receiving the greatest empirical study, refers to the inequitable distribution of groups over an area. Other dimensions include: *Isolation* (degree of potential contact between 2 groups within a city), *concentration* (extent to which minority groups are confined to a compact area within the city), *centralization* (degree to which minorities are clustered around the center of a city), or *clustering* (the extent to which minority neighborhoods are adjacent to one another). Our discussion refers to the general principle of segregation, although it will be an important research endeavor to examine which specific dimensions of segregation are related to environmental health disparities.

Having established a link between race and residence, we now turn to the proximal mechanisms that may account for the relationship between environmental conditions and racial health disparities.

Environmental hazards and pollutants

For space considerations, we assume readers of this journal are familiar with research on environmental justice and environmental health disparities and provide only a brief overview. Environementally relevant disparities are evident in a variety of

outcomes, including asthma, cancer and chemical poisoning (Insitute of Medicine, 1999). Although debated, the main hypothesis explaining these disparities are that disadvantaged communities encounter greater exposure to environmental toxins such as air pollution, pesticides, and lead (Burger et al. 2001; Calderon et al. 1993; Corburn 2002; Fitzgerald et al. 1998; Institute of Medicine 1999; Morello-Frosch 2001; Moses et al. 1993; Northridge and Shepard, 1997; Perera 2003; Pirkle et al. 1998; Woodruff et al. 2003). Mediators of the relationship between toxic exposure and disadvantaged status include the siting of pollution sources (e.g. waste incinerators), illegal dumping, poor enforcement of environmental regulations, inadequate response to community complaints (Maantay 2001; Goldman and Fritton 1994; United Church of Christ 1987; Institute of Medicine 1999; Anderton et al. 1994; Anderton et al. 1997; Bullard 1983; Bullard 1990; Bullard and Wright 1993; Mohai 1992; Perlin 1995; Perlin et al. 1999; Perlin et al. 2001; United Church of Christ 1987; United States General Accounting Office 1983).

Structural factors

Structural factors refer to the historically evolving infrastructure that provides *boundaries for health promotion*. That is, structural factors are constraints that shape how new conditions emerge as salutogens or pathogens in a community. The local economy, for example, is a structural factor that will help determine a community's ability to mobilize resources in order to reject undesirable changes (e.g. introduction of a waste facility) or develop desirable ones (e.g. construction of a park).

We review several of the structural factors that may be especially pertinent to environmental health disparities: the local and national economy, neighborhood physical conditions, land use patterns, and health infrastructure. This is not an exhaustive list, but meant to be illustrative.

One primary effect of residential segregation may be to concentrate disadvantage (Massey and Denton 1993). Compared to whites, minorities are overrepresented in neighborhoods with diminishing and constrained economic opportunities (Jargowsky 1997; Wilson 1987). For example, in Los Angeles in 1990, only 4.9% of blacks lived in high job growth areas, compared to 52.3% of whites (Pastor 2001a). Cutler and Glaeser (1997) reported that a one standard deviation decrease in segregation (13%) would eliminate one-third of the black white differences in education and employment. Thus, segregation may not only concentrate poverty, but it may be partly responsible for the production of poverty among racial minorities (Massey and Denton 1993; Williams and Collins 2001).

There is a clear association between socioeconomic position and health, such that individuals of higher social standing tend to have improved health compared to those of lower standing (Evans 2002; Kaplan et al. 2001; Krieger and Fee 1994; Marmot et al. 1987; Marmot et al. 1998; O'Neill et al. 2003; Williams and Collins 1995). Further, the relationship between socioeconomic position and health holds not only at the individual level, but also at the community (Haan et al. 1987; Kaplan 1996). That is, persons living in poor neighborhoods, even after accounting for their individual socioeconomic characteristics, tend to have worse health outcomes (Diez-Roux et al. 2001; Merkin et al. 2002; Waitzman and Smith 1998; Winkleby and Cubbin 2003).

Neighborhood economic deprivation may compromise health-promoting resources (Diez-Roux et al. 2001). For example, poor and minority neighborhoods tend to have fewer grocery stores with healthy foods (Morland et al. 2002) and fewer pharmacies with needed medications (Morrison et al. 2000). Poor nutrition can increase susceptibility to environmental pollutants by compromising immune function (Beck 1988; Rios et al. 1993). Additionally, disadvantaged neighborhoods are also exposed to greater health hazards, including tobacco and alcohol advertisements, toxic waste incinerators, and air pollution (Morello-Frosch et al. 2002). Finally, economic stress within a community may exacerbate tensions between social groups, magnify workplace stressors, and induce "maladaptive" coping behaviors, such as smoking and alcohol (Brenner 1995). Tobacco and alcohol use can increase susceptibility to environmental toxins that are normally metabolized by impairing host defense (Rios et al. 1993).

In general, racial minorities have lower socioeconomic position compared to whites. Although it is intuitive to hypothesize that disparities in health arise because of socioeconomic differences between racial groups, studies often find that racial disparities persist even after accounting for socioeconomics factors (Williams 1999; Sorlie et al. 1995; Hayward et al. 2000).

Although socioeconomic differences do not completely explain away racial disparities, it is often argued that social class is an important mediator. That is, it is hypothesized that race determines ones economic resources, which in turn determines health (Williams and Collins 1995). Thus, while socioeconomic conditions do not fully account for health disparities, they are a necessary part of the equation.

Neighborhood physical conditions present another structural factor that may contribute to health disparities (Cohen et al. 2003). Minorities are more likely to live in areas with building code violations and neighborhoods with deteriorated housing (Perera et al. 2002; Rosenbaum et al. 1999). In 1999, 3.4% of blacks, 3.8% of Hispanics, and 1.7% of Asian and Pacific Islanders reported living in housing units with severe problems with heating, plumbing, electricity, public areas or maintenance, compared to 1.5% for whites (U.S.Census Bureau 2000). Substandard housing may contribute to a variety of problems, including exposure to toxins, increased risk of injuries from falls and fires, and illness due to ineffective waste disposal and presence of disease vectors (Bashir 2002; Jacobs et al. 2002; Krieger and Higgins 2002; Northridge et al. 2003).

Urban minorities tend to fare worse than their counterparts in rural areas (Geronimus et al. 1999; Geronimus et al. 2001). This may be due in part to land use patterns in urban areas. In Detroit, many minority neighborhoods exist next to highways that expose residents to hazards (Schulz et al. 2002). Sugrue (1996, p.47) argues that "Detroit's highway planners were careful to ensure that construction of new … expressways would only minimally disrupt middle-class residential areas, but they had little such concern for black neighborhoods." Similarly, New York City rezoned its neighborhoods between 1961 to 1998 so as increase manufacturing zones in areas with higher minority populations and to decrease those zones in areas with fewer minorities (Mantaay, 2001). Those rezoning efforts led to higher concentration of industrial burden within manufacturing designated areas. Further, some policies that appear neutral *prima facie* may result in adverse impact on already disadvantaged communities, as in

the example of emissions trading systems and their potential to create pollution "hotspots"(Solomon and Lee 2000; Schmidt 2001).

Health infrastructure may also be associated with race. Minorities tend to reside in areas with a lower physician per population ratio and medication supply (Morrison et al. 2000; Rosenbaum et al. 1999; Schulz et al. 2002). Community hospitals are more likely to close in urban minority communities (Whiteis 1992). These findings suggest that segregated communities face structural disadvantages in the provision of health services.

Because so many different structural forces appear to confer disadvantage among minority communities, some scholars have suggested that they continue a history of institutionalized discrimination against minorities (Jones, 2000; Williams, 2001; Krieger et al., 1993; Feagin, 2003; Gee, 2002; Squires, 1994; Massey and Denton, 1993). This discrimination may not have a purposeful intent, but still may confer adverse impact.

Community stressors:

Community stress theory derives from a century of research on the stress process among individuals (Lazarus and Folkman 1984; McEwen 1998; Selye 1936; Steptoe and Feldman 2001; Schulz et al. 2000). *Stress* is a state of activation of physical and psychological readiness to act in order to help an organism survive external threats. *Stressors* are the factors that produce stress, and include phenomena like crime (Morenoff et al. 2001 ), noise (Babisch et al. 2001; Ouis 2001), traffic (Gee and Takeuchi, 2004), litter, density and residential crowding (Fleming, 1987; Evans,

1993). Stressors can result directly from environmental hazards, including technological and natural disasters (Baum,et al, 1983; Brown, 2002).

*Health effects of stress*

Stressors can trigger the sympatho-adrenal system, hallmarked by rapid release of adrenalin and noradrenalin, leading to various "fight or flight" responses, including arousal, bronchodilation, tachycardia, and increased blood pressure. The hypothalamic-pituitary-adrenal system is also activated, signified by release of corticotrophin releasing factor, adrenocorticotropic hormone and cortisol. These glucocorticoids have several metabolic and psychological effects, including the mobilization of energy reserves, suppression of the immune system, and heightened vigilance. Chronic activation of the stress system is believed to lead to allostatic load, defined as the "wear and tear" on organ systems resulting from stress (McEwen 1998; Pope, et al., 1999). A full discussion of the biology of stress is beyond the scope of this paper, but can be found in several accounts (Brunner 2000; Hadley 1992; McEwen 1998).

The key point is that stressors can cause illness by weakening the body's ability to defend against external challenges. As an example, Cohen and colleagues (Cohen et al. 1991) asked volunteers to self-rate their levels of stress, then randomized them to received nasal drops containing either placebo or respiratory viruses. Rates of respiratory infection and clinically diagnosed colds followed a positive dose-response with level of psychological stress. Findings from this controlled experiment were further unaffected by controls for a variety of factors (e.g. allergic status).

Intriguingly, some evidence suggests that stress may influence the internal does of a given toxin. This is because stress may: (1) increase the absorption of toxins into the body through increased respiration, perspiration, and consumption (Gordon 2003); (2) compromise host defense systems (McEwen 1998); (3) and directly cause illness, which in turn, may lead to an amplification loop, whereby sick individuals are less likely to cope with environmental toxins (Rios et al. 1993). Stress may induce or unmask a latent effect of a toxicant, possibly altering basal levels of neurofunctioning and shifting the threshold for neurotoxicity (U.S. Department of Health and Human Services 1995).

Two factors are purported to determine individual response to stress: how one appraises the situation and their general state of physical health (Lazarus and Folkman 1984; McEwen 1998). Coping resources, such social support, help determine the extent to which a stressor is perceived as a threat and subsequent health (Israel et al. 2002). For example, workers with high levels of job strain and low levels of co-worker support have higher risk of cardiovascular disease than those with similar levels of strain and more support (Johnson et al. 1996). Additionally, physical illness will impair an individual's ability to respond to stressors. Individual stress and coping have macro level analogues, community stressors and neighborhood resources.

*Types of Community stressors*

Community stressors can be categorized into two major types, physical and psychosocial. Physical conditions, including noise, temperature, humidity, barometric/water pressure, visible light, geomagnetism, radiation and particulate matter may contribute to stress (see Gordon, 2003 for review). These stressors can induce a

physiological response that makes the body more susceptible to illness. Heat stress, for example, induces sweating and increased skin blood flow, which in turn can facilitate the transcutaneous absorption of pesticides (Chang et al. 1994; Funckes et al. 1963; Wester et al. 1996). Individuals subject to ambient noise have higher levels noradrenalin, a stress biomarker (Babisch et al. 2001). In a natural experiment, Evans and colleagues (Evans et al. 1998) found that the chronic exposure to aircraft noise elevated resting blood pressure, norepinephrine and epinephrine biomarker levels, and decreased self-reported quality of life over a two year period.

Psychosocial conditions -- including crowding, social disorganization, racial discrimination, fear, and economic deprivation – may also be sources of stress (Krieger and Higgins 2002; Macintyre et al. 2002; Schulz et al. 2000; Steptoe and Feldman 2001). One stressor that has received extensive attention is fear of crime (Morenoff 2003; Warr and Ellison 2000). Minority neighborhoods tend to have higher crime rates, which may contribute to health disparities. Perceptions of crime and disorder within an individual's community has been associated with numerous outcomes, including anxiety depression, post-traumatic stress disorder, and substance use (Aneshensel and Sucoff 1996; Cutrona et al. 2000; Fick and Thomas 1995; Geis and Ross 1998; Ross et al. 2000; Ross and Jang 2000). Morenoff (2003) found that the neighborhood violent crime rate was one of the "most robust" environmental predictors of infant birth weight, after controlling for both individual (e.g. smoking during pregnancy) and neighborhood characteristics (e.g. % poor families).

Physical and psychosocial stressors may interact with one another, as seen with natural and technological disasters (Arata et al. 2000; Baum et al. 1983; Kaniasty and

Norris 2000). For example, the trauma of the Love Canal incident in New York resulted from both the chemical hazards as well as the public perceptions (Edelstein and Wandersman 1987; Gibs 1983, Holden 1980). Further, the relationship between environmental and subjective stressors occurs not only for highly salient events, but also for everyday events. Gee and Takeuchi (2004), using multilevel models, reported that persons perceiving stress due to automobile traffic had greater psychological distress and lowered general health status than those perceiving less stress. However, these outcomes were worst for persons perceiving high stress *and* living in high traffic areas.

*Racial disparities in exposure to stressors*

Further, there are racial disparities in the burden of stressors that accumulate over the life course (Geronimus et al. 2001; Holland et al. 2000; Williams, 1997a; Krieger, et al., 1993; Jones, 2000). Some have called this racially differential burden of cumulative stress the "weathering hypothesis" (Geronimus, 1996 Astone et al. 2002). One of the most prominent stressors may be racial discrimination (Gee 2002; Krieger and Sidney 1996; LaVeist et al. 2000; Williams et al. 1997a; Williams and Neighbors 2001). Because racial discrimination has profoundly shaped the experiences of racial groups, discrimination may be among the factors that shape health disparities. Evidence suggests that racial discrimination still occurs in the present day, especially in structurally important domains such as housing, education and employment (Essed 1992; Feagin 2000). Audit studies send a white and a minority prospective tester with identical portfolios (e.g. similar income and job titles) to assess a given housing market.

These audits have consistently found that whites are favored over minorities. Hispanics, for example, are more likely to be quoted a higher rent for a given unit than their white counterparts (Turner and Skidmore 2001). Other studies have shown that minorities are more likely to face discrimination in applying for a job (Kirschenman and Neckerman 1991), or shopping (Lee 2000). Further, discriminatory treatment within the health care system also might contribute to disparities (Krieger 1999) . Minorities appear to have longer waiting times for kidney transplants (Eggers 1995; Klassen et al. 2002), liver transplants (Kjellstrand 1988; Young and Gaston 2000), and report less satisfaction with their medical visits (Cooper-Patrick et al. 1999; Saha et al. 2003). The Institute of Medicine's (2002) review concluded:

> "Racial and ethnic minorities tend to receive a lower quality of healthcare than non-minorities, even when access-related factors, such as patients' insurance status and income are controlled… the study committee found evidence that stereotyping, biases, and uncertainty on the part of healthcare providers can all contribute to unequal treatment…."

Stress from discrimination may lead to illness. Kessler and colleagues (1999) have suggested, "the conjunction of high prevalence and strong impact would mean that discrimination is among the most important of all the stressful experiences that have been implicated as causes of mental health problems." Studies have reported that stress due to racial discrimination is associated with high blood pressure (Krieger and Sidney 1996), mental health (Dion et al. 1992; Gee 2002; Kessler et al. 1999; Kuo 1976; Williams et al. 1997b), and alcohol consumption (Yen et al. 1999).

Neighborhood Resources

Although a common argument is that segregation is harmful to the health of minorities, there is some indication that segregation may have a counterbalancing effect by concentrating social resources, such as black political power (LaVeist 1993). Others have reported that the clustering of ethnic groups may build a sense of collective identity that helps mitigate trauma (Mazumdar et al. 2000). Thus, supportive social relationships within minority communities may help promote health and well-being and ameliorate the effects of community risks. Our view is that segregation concentrates both risks and resources. It is not a matter of whether segregation is either "bad" or "good," but to what degree the negative effects of segregation outweigh positive effects.

Neighborhood resources buffer community stressors (Kretzman and McKnight 1993). Generally, these resources have been conceptualized in terms of relationships between residents, including: social cohesion, social capital, psychological sense of community, informal social control, and community empowerment (Berkman and Glass 2000; Kawachi et al. 1999; Ross and Jang 2000; Sampson et al. 1997). *Social cohesion* is defined as the "extent of connectedness and solidarity among groups in society (Kawachi and Berkman, 2000, p.175)." Essentially, a community with a high degree of social cohesion has strong social ties between members and minimal conflict. *Social capital* can be considered a type of resource that emerges from socially cohesive groups that facilitates collective action. These resources include norms of reciprocity, aid and interpersonal trust.

Collective efficacy, defined as "mutual trust and willingness to intervene for the common good," (Sampson et al. 1997) may mediate the adverse effects of concentrated disadvantage and fear (Ross and Jang, 2000). Pastor and colleagues (Pastor 2001b)

suggested that social capital was stronger in communities with less “ethnic churning,” referring to the replacement of one minority group with another within a community. They argued that ethnic churning may “weaken the usual social bonds constituted by race and make an area more susceptible to siting of noxious land uses.” Their data indicated that ethnic churning in Los Angeles was associated with the siting of hazardous waste storage and disposal facilities over a two decade period, after adjusting for economic factors.

Another potential resource is residents’ ability to control their environment, which may mitigate community problems in two ways. First, empowered communities may be able to protect themselves from the instruction of new hazards and eliminate extant ones (Bullard and Wright 1993; Lee 1993; Morello-Frosch et al. 2002; Phoenix 1993; Rich et al. 1995; Zimmerman 2000). These communities may also be able to control the political arena that shapes their health beyond the effect of environmental pollutants. Black political participation, defined by the presence of African American legislators, has been associated with lower mortality rates in African American communities (LaVeist 1993). This is possibly due to a higher preponderance among African American communities to provide a wider range of social services compared to white communities (Schneider and Logan 1982). Second, control *per se* may be an important factor determining stress and health. Workers with greater control over their work process have lower risk of cardiovascular disease than workers with less control (Karasek and Theorell 1990; Kuper and Marmot 2003; Landsbergis et al. 1997). Further, collective control by workers and their unions may also provide health benefits (Johnson, 1989; Sorenson, et al, 2004).

## Community Stress

The cumulation of environmental pollutants, structural process, community stressor and neighborhood resources is community stress. Community stress is a state of ecological vulnerability. Community resources help buffer community stressors and protect against environmental exposure, but when resources are inadequate, community stress arises. Structural factors constrain the limits of resources and stressors.

Although several factors cross the threshold from "community" to "individual," we focus on the intersection between community stress and individual stress. In particular, community stress may itself lead to individual stressors. These individual stressors may in turn, lead to individual stress and subsequent illness. The terrorist attacks of September 11, 2001 provides and an extreme example of how community stress can translate to individuals. The attack was a threat to the American "community." Although most citizens were not close to the epicenter, many individuals across the United States felt some measure of distress from the attack.(Schuster et al. 2001; Schlenger et al. 2002)

## Future Directions

This framework is meant to stimulate dialogue between environmental and social scientists. Several avenues for future work are suggested. First and foremost, while several components within the framework have undergone extensive study, such as between individual stress and health, relatively little work has attempted to integrate the elements as a whole. Studies are just beginning to consider the connections between

factors at multiple levels, such as between community stress, individual stress, and health. Future work should continue to test the components of the framework and incorporate multi-level modeling (Raudenbush and Bryk 2002). Longitudinal studies will be necessary to establish the temporal ordering between variables.

Second, public health should more seriously consider the role that residential segregation plays in the production of health disparities. Several lines of inquiry are possible with regards to segregation alone. For example, how might environmental risk perception play in maintaining segregation? Are certain dimensions of segregation more important than others? Are the mechanisms linking segregation to health all negative, or might there be some health promoting pathways, such as in the clustering of cultural resources? What are the forms of segregation outside of the U.S.A. and are the mechanisms similar? Does the relationship between segregation and health generalize to all ethnic groups?

Third, we hope that this framework will encourage the environmental justice movement to expand the notion of "environmental hazards" to include community stressors. Are minority communities more likely to receive the siting of workplaces with high job strain (Karaek and Theorell, 1990)? Do differences in community stress lead to the "weathering" (Geronimus 1996) of minority communities compared to whites? This means not only examining the main effects of stress and toxins, but also examining whether psychosocial stress may potentiate (i.e. amplify) the effects of toxins on the body.

Fourth, research should not only examine the relationship between minority communities and exposures, but also, study how the structural conditions of

communities may confer additional vulnerability. Disadvantaged communities may be more vulnerable to exposure to environmental hazards because structural conditions, such as substandard housing, may render them more likely to be exposed than counterparts in more advantaged communities equally distant to these hazards. That is, do minority communities have less protection against a given level of exposure and do these disparities in protection result from differential social policy?

## Conclusion

Our work has implications for environmental justice by suggesting that exposure to physical and chemical hazards is only one route whereby neighborhoods impact the health of racial minorities. Health promotion may require policies and interventions aimed at eliminating environmental toxins, fostering community resources, and reducing social stressors. Reduction of the *gap* in health between advantaged and disadvantaged groups, however, may require interventions targeted at eliminating the gap in advantages themselves.

We emphasize racial differences in *exposure to* stress, rather than racial differences in *response to* stress. The former conceptualization emphasizes interventions on macro social policy (e.g. housing policy), whereas the latter perspective emphasizes interventions at the micro level (e.g. psychological counseling or pharmacological agents). While micro approaches are useful, one disadvantage is that individual interventions require tremendous resources in order to manifest outcomes at the population level (and hence, reduce group differences) and further, are less efficient because interventions must be reapplied to each new birth cohort. However, policy

level changes that target socially-produced stressors may prove a promising way to improve the public's health.

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Table 1. Segregation of Ethnic Minorities to Whites, United States, 1980-2000*

| | 1980 | 1990 | 2000 |
|---|---|---|---|
| American Indian | 37.3 | 36.8 | 33.3 |
| African Americans | 72.7 | 67.8 | 64.0 |
| Asians & Pacific Islanders | 40.5 | 41.2 | 41.1 |
| Hispanics | 50.2 | 50.0 | 50.9 |

* Segregation is measured with the Index of Dissimilarity. The Index measures the evenness of groups over space and can be interpreted as the percent of a particular group who would have to move in order integrate the two groups over the region as a while. For example, in the year 2000, 64% of all African Americans (or whites) would have to another census tract in order to integrate all metropolitan areas nationwide. Data are adapted from the U.S. Census (2003) (http://www.census.gov/hhes/www/housing/resseg/tab7-1.html)

# Figure Legends

Figure 1 Exposure-Disease-Stress Model for Environmental Health Disparities

**Figure 1. Exposure-Disease-Stress Framework for Environmental Health Disparities**